CLINICAL SCIENCE

Exercise-induced improvements in cardiorespiratory fitness and heart rate response to exercise are impaired in overweight/obese postmenopausal women

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OBJECTIVE: The purpose of this study was to compare the heart rate response to exercise and the exercise-induced improvements in muscle strength, cardiorespiratory fitness and heart rate response between normal-weight and overweight/obese postmenopausal women.

METHODS: Sedentary women (n = 155) were divided into normal-weight (n = 79; BMI <25 kg/m^2; 58.3 $\pm$ 8.6 years) and overweight/obese (n = 76; BMI ≥ 25 kg/m^2; 58.3 $\pm$ 8.6 years) groups, and have their 1-repetition maximum strength (adjusted for body mass), cardiorespiratory fitness and heart rate response to a graded exercise test compared before and after 12 months of a three times-per-week exercise-training program.

RESULTS: Overweight/obese women displayed decreased upper and lower extremity muscle strengths, decreased cardiorespiratory fitness, and lower peak and reserve heart rates compared to normal-weight women. After follow-up, both groups improved their upper (32.9% and 41.5% in normal-weight and overweight/obese women, respectively) and lower extremity(49.5% and 47.8% in normal-weight and overweight/obese women, respectively) muscle strength. However, only normal-weight women improved their cardiorespiratory fitness (6.6%) and recovery heart rate (5 bpm). Resting, reserve and peak heart rates did not change in either group.

CONCLUSIONS: Overweight/obese women displayed impaired heart rate response to exercise. Both groups improved muscle strength, but only normal-weight women improved cardiorespiratory fitness and heart rate response to exercise. These results suggest that exercise-induced improvements in cardiorespiratory fitness and heart rate response to exercise may be impaired in overweight/obese postmenopausal women.

KEYWORDS: Exercise; Obesity; Cardiorespiratory fitness; Heart rate; Muscle strength.

Ciolac EG and Greve JMD. Exercise-induced improvements in cardiorespiratory fitness and heart rate response to exercise are impaired in overweight/obese postmenopausal women. Clinics. 2011;66(4):583-589.

Received for publication on November 10, 2010; **First review completed on** December 25, 2010; **Accepted for publication on** January 10, 2011

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INTRODUCTION

Obesity is an increasingly prevalent health problem in developed and developing countries and poses a significant challenge to both individual and public health.[1,2] Obesity is strongly linked to a higher risk for cardiovascular disease, diabetes, several cancers, and other chronic conditions.[3,4]

Poor cardiorespiratory fitness (CRF) and muscle strength and abnormalities in the autonomic nervous system (ANS) are important predictive factors of morbidity and mortality associated with obesity.[5-9] ANS alterations are indicated by decreased baroreflex sensitivity[9-11] and lower heart rate (HR) variability[8,11] in obese subjects compared with normal-weight individuals. The standard graded exercise test (GXT) is an easy and inexpensive tool that provides a wealth of information on the interactions between the ANS and the cardiovascular system at various phases of rest, exercise and recovery.[12-14] Moreover, the HR response to a GXT is a powerful predictor for cardiovascular mortality and morbidity.[12,13] However, there is little information regarding the HR response to GXT in overweight and obese subjects, especially in postmenopausal women.

Physical exercise is a key strategy in the management of obesity. Numerous health-related benefits have been observed in overweight and obese people who participate in exercise training programs, even in those without significant weight loss.[15,16] For example, CRF improves in overweight and obese subjects following exercise training programs.[17-19] However, changes in skeletal muscle structure have been observed in overweight and obese individuals compared to normal-weight subjects, which may contribute to reduced CRF and muscle strength improvements in response to exercise training in overweight/obese subjects. These differences include a lower percentage of type I muscle

fibers,[20] impaired muscle oxidative capacity[19,20] and microvascular function,[21] inability to increase fat oxidation during β-adrenergic stimulation and exercise,[22] and increased intramuscular lipid storage.[20,22] In addition, excess adipose tissue releases a variety of neurohormones and cytokines that blunt insulin-stimulated glucose uptake by muscle.[20] In line with these alterations, overweight and obese individuals have a lower tolerance to high-intensity exercise[23] and higher perceived exertion at a given exercise intensity,[24] and they are more willing to participate in exercise for longer durations at a lower exercise intensity than normal-weight individuals.[25] Moreover, studies comparing the muscle strength response to exercise between overweight/obese and normal-weight individuals have shown discrepant results.[26-29]

The obesity-related abnormalities of the ANS[9-11] could also contribute to an impaired exercise-induced adaptation of the HR response to GXT in overweight and obese subjects. However, little is known regarding exercise-induced adaptations in the CRF and HR responses to GTX in overweight and obese subjects. Therefore, the aims of the present study were the following: (1) to compare the HR response to GXT between normal-weight and overweight/obese postmenopausal women and (2) to compare the effects of exercise training on the CRF and HR responses to GXT and muscle strength between normal-weight and overweight/obese postmenopausal women.

METHODS

Population

The study population consisted of non-smoking, physically inactive, postmenopausal women, aged 45 to 69 years. Participants were referred for pre-participation physical exercise screening, and they underwent GXT and 1-repetition maximum strength (1-RM) test between August 1, 2004 and August 31, 2008 before participating in the Cardiovascular and Muscular Fitness Program at the Laboratory of Kinesiology at the Institute of Orthopedics and Traumatology, Medical School, University of Sao Paulo. A structured history, medical record review and physical evaluation were performed before the exercise testing to document symptoms, history of chronic diseases, current medication, cardiac risk factors, and cardiac events and procedures. All women with musculoskeletal limitations to physical exercise, uncontrolled cardiovascular or metabolic disease, insulin-dependent diabetes, chronic psychological disorders, cardiac disease or the use of any drug that could potentially influence muscular or cardiovascular response to exercise were excluded from the study. Women who did not complete at least 75% of the exercise sessions were excluded from the final analyses (Figure 1). The ethics committee at the Institute of Orthopedics and Traumatology, Medical School, University of Sao Paulo approved all procedures, and the study participants read a detailed description of the protocol and provided written informed consent.

Graded Exercise Test (GXT)

The subjects performed a maximal, symptom-limited treadmill test using a Heck-modified protocol at controlled room temperature (20-23℃) before and after 12 months of follow-up. The test consisted of 1 minute of rest standing; 1 minute each of warm-up at 2.4, 3.6 and 4.8 km/h; increases of 1.2 km/h every 1.5 minutes until volitional fatigue in the absence of symptoms or other indicators of ischemia[30]; 2 minutes of recovery at 2.4 km/h; and 1 minute of recovery standing. GXT intensity was measured in metabolic equivalents (METs) estimated from treadmill speed and grade using a standardized equation.[31] Cardiac rhythm was continuously monitored by electrocardiogram in 12 derivations and recorded for 10 seconds at the end of rest, end of each warm-up and exercise stage, and at the end of each minute of recovery phase. Arterial blood pressure (BP) was measured at the end of rest, the end of each exercise stage and at the end of each minute of recovery phase. Indirect arm-cuff sphygmomanometry was to determine BP. Medications were not changed or stopped before testing. Subjects were told to avoid caffeine and alcoholic beverages for 24 hours and to have a light meal up to 2 hours before the GXT. Age-predicted maximal HR was determined using a standardized equation.[32] Reserve HR ($HR_{RESERVE}$) was defined as the difference between HR at the end of the GXT (HR_{PEAK}) and the resting HR ($HR_{RESTING}$). Recovery HR ($HR_{RECOVERY}$) was defined as the difference between HR_{PEAK} and the HR at the end of first minute of recovery phase.

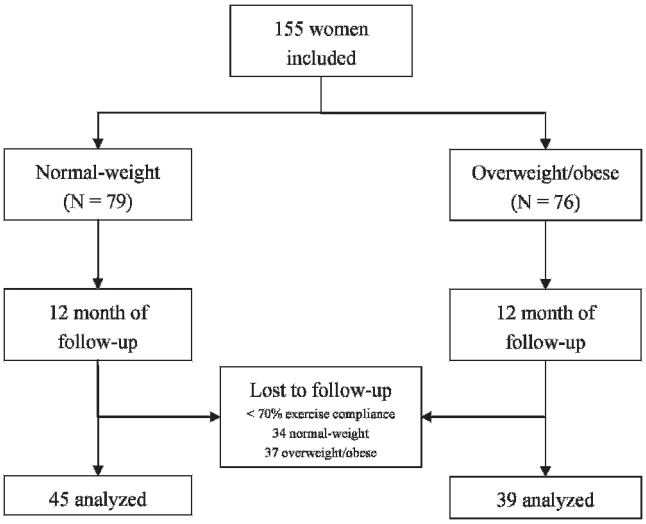

Figure 1 - Flow diagram of the study design.

Strength Test

To determine muscle strength and initial workload for each resistance exercise, the 1-repetition maximum 1-RM test was performed after four familiarization bouts and 2-5 days after the last exercise session as previously described.[33,34] In brief, the 1-RM test was performed in bench press, leg press, seated row, knee extension, shoulder press, knee curl, biceps curl, calf raise, triceps push-down and abdominal exercises using the same weight-lifting machines and free-weight dumbbells that were used for training (Multiflex©, Biodelta Inc, Brazil). Tests were conducted following the exercise order described above (after proper warm-up). The 1-RM workload was defined as the maximum weight that could be moved once through the full range of motion with proper form and without performing the Valsalva maneuver. All tests were conducted by the same investigator before and after the exercise-training period. The muscle strength data were normalized for body mass with the allometric method (strength [kg] x body mass $[kg]^{-0.67}$)[35]. The sum of the

normalized upper extremity (bench press, seated row, shoulder press, biceps curl, triceps push-down and abdominal) and lower extremity (leg press, knee extension, knee curl, and calf raise) data were used for between-group comparisons. The intraclass correlation for the 1-RM test-retest measures was 0.983 (95% confidence interval = 0.964-0.997).

Exercise Training Program

The exercise training program consisted of supervised and unsupervised exercise sessions. The unsupervised exercise sessions were held once per week and consisted of 40 minutes of walking at an intensity between 11 and 14 on the perceived exertion scale.[36] The supervised exercise sessions, which were monitored by an exercise specialist, were conducted twice per week and consisted of aerobic, resistance and stretching exercises. Aerobic exercise was performed at the beginning of each exercise session and consisted of 20 minutes on a cycle ergometer or walking at 60-75% of the $HR_{RESERVE}$ heart rate.[37] Resistance exercises consisted of 2-3 sets of 8-12 repetitions each at 60-80% of the 1-RM, with all of the exercises described in the 1-RM test being performed. Participants stretched at the end of each session by reaching and sustaining the maximum range of motion for 20 seconds in 10 exercises designed to target the major muscle groups. To promote a sufficient workload and maintain improvements throughout the 12 months of training, the aerobic and resistance exercise intensities were increased by 5% and 1-5 kg (5-10%), respectively, whenever an adaptation occurred. Aerobic exercise adaptation was defined as an exercise HR lower than 70% of the subject's reserve HR for one exercise session. Resistance exercise adaptation was defined as when 2 sets of 12 repetitions were performed with proper form and the Valsalva maneuver was avoided for two consecutive exercise sessions.

Statistical Methods

All data (reported as mean $\pm$ standard deviation) were analyzed with subjects divided into the normal-weight (BMI $<25\ kg/m^2$) or overweight/obese (BMI $\geq 25\ kg/m^2$) groups. Because several studies have suggested that other metabolic abnormalities such as high hemoglobin A_{1c} levels and fasting hyperglycemia may be associated with ANS abnormalities rather than with obesity,[38,39] the subjects were further divided into subgroups according to the presence of comorbidities (insulin resistance, diabetes, arterial hypertension, or dyslipidemia).

The SPSS 12.0 statistical program for Windows (SPSS Inc., Chicago, IL, USA) was used for statistical analyses. The Kolmogorov-Smirnov test was applied to ensure a Gaussian distribution of the data. Student's *t* tests for unpaired variables and Kruskal-Wallis tests were used to determine differences between groups for parametric and nonparametric data, respectively. When the subjects were subdivided according the presence of comorbidities, the between-group differences were determined by a one-way analysis of variance (ANOVA), and the *post-hoc* Bonferroni's test was used to identify significant differences indicated by the ANOVA tests. A two-way ANOVA with repeated measurements (group vs. time) was used to indicate inter- and intra-group differences in the studied variables. A Bonferroni *post-hoc* analysis was used to determine significant differences that were determined by the two-way ANOVA. Statistical significance was set at $P<0.05$.

RESULTS

A total of 155 postmenopausal women (76 of whom were overweight or obese) were included in the study. The prevalence of comorbidities, such as hypertension, diabetes mellitus and dyslipidemia, and the use of medications to treat these conditions were greater in overweight/obese women compared to normal-weight women (Table 1). Baseline data for the GXT and 1-RM tests are displayed in Table 2. Overweight/obese women also displayed lower CRF (-11.1% and -10.0% for METs and exercise time, respectively; $P<0.01$) and muscle strength (-12.9% and -13.7% for upper and lower extremities, respectively; $P<0.001$) compared to normal-weight women. Resting, recovery and absolute reserve HRs were not significantly different between groups; however, overweight/obese women displayed lower absolute and relative (% of predicted) HR_{PEAK} values and lower relative $HR_{RESERVE}$ values than normal-weight women ($P<0.05$). Overweight/obese women also displayed higher resting, peak and recovery systolic and diastolic BPs than normal-weight women ($P<0.05$). Women subdivided into groups based on the presence or absence of comorbidities showed the same pattern as observed in the entire population (Table 3).

Although 155 women were included in the study, only 84 (39 of whom were overweight or obese) completed at least 75% of the exercise sessions and were included in the final analyses. No baseline differences were observed in muscle strength, CRF or HR response to exercise between subjects with or without comorbidities in the same BMI group. Therefore, the effects of exercise training on these variables were analyzed on the subjects with or without comorbidities in a single BMI group (normal-weight or overweight/obese). There were no statistical differences between women who completed or women who did not complete the study at baseline. Neither BMI nor waist circumference were different after 12 months of follow-up in the normal-weight ($BMI = 22.3 \pm 1.6$ *vs.* 22.1 ± 1.6, $P = 0.269$; waist circumference $= 80.9 \pm 7.6$ *vs.* 78.7 ± 7.6, $P = 0.104$) or overweight/obese

Table 1 - Demographic and clinical characteristics of the study population.

Characteristics	Normal-weight (n = 79)	Overweight/ Obese (n = 76)	P
Age, year	50.3 ± 8.6	50.6 ± 9.1	0.83
Height, cm	159 ± 7	159 ± 6	0.78
Weight, kg	56.8 ± 6.2	74.4 ± 11.7	<0.001
BMI	22.5 ± 1.6	29.3 ± 3.5	<0.001
Waist circumference, cm	81.3 ± 6.4	97.2 ± 10.5	<0.001
Systolic BP, mmHg	107.1 ± 13.6	116.1 ± 18.3	0.004
Diastolic BP, mmHg	68.0 ± 9.1	75.3 ± 18.3	<0.001
Hypertension, n (%)	11 (14.3)	24 (31.6)	<0.001
Diabetes, n (%)	2 (2.5)	10 (13.2)	0.03
Dyslipidemia, n (%)	5 (6.5)	11 (14.5)	0.04
Hormone Replacement Therapy, n (%)	2 (2.5)	2 (2.6)	0.97
Current Medication			
CCB, n (%)	3 (3.9)	2 (2.6)	0.75
ACE-I, n (%)	6 (7.8)	13 (17.1)	0.02
Diuretics, n (%)	7 (9.1)	17 (22.4)	0.009
Metformin, n (%)	2 (2.5)	10 (13,2)	0.01
Statins, n (%)	5 (6.5)	11 (14.5)	0.03

CCB, calcium channel blocker; ACE-I, angiotensin-converting enzyme inhibitor. BMI, body mass index.

Table 2 - Baseline muscle strength, cardiorespiratory fitness, heart rate and blood pressure response to exercise.

Characteristics	Normal-weight (n = 79)	Overweight/ Obese (n = 76)	P
1- RM Test •			
Upper extremity, $kg \cdot kg^{-0.67}$	9.40 ± 1.61	8.21 ± 1.84	<0.001
Lower extremity, $kg \cdot kg^{-0.67}$	10.26 ± 2.31	8.85 ± 2.13	<0.001
GXT			
Exercise capacity, METs	9.88 ± 1.86	8.79 ± 1.34	0.007
Exercise time, min	9.51 ± 1.36	8.55 ± 1.42	<0.001
$HR_{RESTING}$, bpm	85.0 ± 11.9	84.0 ± 12.7	0.439
HR_{PEAK}, bpm	180.8 ± 9.3	172.0 ± 14.2	0.022
% of predicted HR_{PEAK}	104.6 ± 4.7	101.2 ± 5.0	0.012
$HR_{RESERVE}$, bpm	95.2 ± 13.8	91.0 ± 16.5	0.244
% of predicted $HR_{RESERVE}$	109.7 ± 9.7	102.3 ± 11.3	0.011
$HR_{RECOVERY}$, beats	19.9 ± 6.9	18.0 ± 7.5	0.098
Blood Pressure			
$SBP_{RESTING}$, mmHg	108.8 ± 14.3	118.1 ± 18.3	0.01
$DBP_{RESTING}$, mmHg	69.3 ± 9.1	76.3 ± 13.8	<0.001
SBP_{PEAK}, mmHg	149.4 ± 15.9	159.9 ± 21.8	0.01
DBP_{PEAK}, mmHg	66.1 ± 12.1	71.7 ± 13.5	0.04
$SPB_{RECOVERY}$, mmHg	136.4 ± 20.0	147.9 ± 23.9	<0.001
$DBP_{RECOVERY}$, mmHg	66.2 ± 11.6	71.6 ± 11.4	0.03

•Adjusted for body mass. SBP, systolic blood pressure; DBP, diastolic blood pressure. 1-RM, 1-repetition maximum strength ;GXT, graded exercise test; HR, heart rate; SBP, systolic blood pressure; DBP, diastolic blood pressure.

(BMI = 29.2 ± 3.0 *vs.* 29 ± 2.9, P = 0.419; waist circumference = 97.4 ± 10.6 *vs.* 95.8 ± 9.3, P = 0.44) groups.

Data from the GXT and 1-RM tests of the women completing the exercise training program before and after the 12-month follow-up are presented in Table 4. Both groups improved upper (39.4%) and lower (49.6%) extremity muscle strengths similarly after exercise training (P<0.001). However, only the normal-weight women improved CRF significantly (METs = 6.6 ± 2.2%; exercise time = 9.8 ± 3.8%; P<0.05). Absolute and relative HR_{PEAK} and $HR_{RESERVE}$ values did not change significantly in either group after exercise training; however, $HR_{RECOVERY}$ improved (14.1%) in the normal-weight (P<0.05) group but not in the overweight/obese group. Both groups displayed lower $HR_{RESTING}$ values, and the overweight/obese women had lower resting and peak BPs in response to GXT, but these reductions were not statistically significant.

DISCUSSION

The management of cardiorespiratory function in overweight/obese women is one of the challenges facing the 21st century.[40] Our study found that overweight/obese postmenopausal women display an impaired HR response to exercise and an impairment in the exercise-induced improvement in CRF and $HR_{RECOVERY}$ compared to normal-weight women. However, no differences were observed between the groups for exercise-induced improvements in upper and lower extremity muscle strengths.

Muscle strength and CRF are reduced in overweight/obese individuals compared with those of a normal weight.[5-7] In this study, the reduced baseline CRF (12%) and upper (9.4%) and lower (9.8%) extremity strengths observed in overweight/obese than normal-weight postmenopausal women are in agreement with previous studies. Skeletal muscle differences, such as a lower percentage of type I muscle fibers,[20] impaired muscle oxidative capacity[19,20] and microvascular function,[21] inability to increase fat oxidation during β-adrenergic stimulation and exercise,[22] and increased intramuscular lipid storage[20,22], have been postulated to impair CRF and muscle strength in overweight/obese subjects. However, it was not known whether the cardiovascular and skeletal muscle

Table 3 - Baseline muscle strength, cardiorespiratory fitness, heart rate and blood pressure responses to exercise in women with or without comorbidities. 1-repetition maximum strength test was adjusted for body mass.

	Subjects Without Comorbidities		Subjects With Comorbidities	
Characteristics	Normal-weight (n = 65)	Overweight/obese (n = 45)	Normal-weight (n = 14)	Overweight/obese (n = 31)
1- RM Test •				
Upper extremity, $kg.kg^{-0.67}$	9.46 ± 1.44	8.30 ± 1.53 ***	8.72 ± 1.93	7.67 ± 1.81 ***
Lower extremity, $kg.kg^{-0.67}$	10.36 ± 2.24	9.01 ± 2.07 ***	9.20 ± 1.99	8.00 ± 2.06 ***
GXT				
Exercise capacity, METs	9.95 ± 1.49	9.01 ± 1.15 **	9.65 ± 0.89	8.17 ± 1.38 **
Exercise time, min	9.68 ± 1.25	8.71 ± 1.29 **	8.81 ± 1.12	7.77 ± 1.31 **
$HR_{RESTING}$, bpm	84.8 ± 10.3	83.5 ± 13.3	85.5 ± 15.5	84.1 ± 11.0
HR_{PEAK}, bpm	181.0 ± 9.1	173.3 ± 5.0 *	176.7 ± 9.1	170.5 ± 14.2 *
% of predicted HR_{PEAK}	104.9 ± 4.6	101.2 ± 5.3 **	104.3 ± 4.4	101.1 ± 4.5 *
$HR_{RESERVE}$, bpm	96.8 ± 12.1	93.7 ± 17.7	91.5 ± 13.6	88.4 ± 15.7 *
% of predicted $HR_{RESERVE}$	110.0 ± 9.7	102.6 ± 10.2 **	109.3 ± 13.1	101.9 ± 12.7 **
$HR_{RECOVERY}$, beats	20.7 ± 5.9	17.5 ± 6.5	17.2 ± 7.4	19.4 ± 9.4
Blood Pressure				
$SBP_{RESTING}$, mmHg	106.2 ± 12.9	113.4 ± 16.9 **	113.0 ± 15.1 [a]	126.3 ± 19.4 **[a]
$DBP_{RESTING}$, mmHg	67.6 ± 8.7	72.4 ± 9.7 **	72.9 ± 11.0 [a]	85.1 ± 17.1 **[a]
SBP_{PEAK}, mmHg	147.5 ± 13.8	154.9 ± 16.7 **	150.4 ± 19.4 [a]	168.5 ± 20.3 **[a]
DBP_{PEAK}, mmHg	63.9 ± 10.4	67.7 ± 11.9 **	70.3 ± 10.9 [a]	77.6 ± 11.3 **[a]
$SPB_{RECOVERY}$, mmHg	131.6 ± 15.3	142.9 ± 19.6 **	146.4 ± 15.4 [a]	156.1 ± 19.9 **[a]
$DBP_{RECOVERY}$, mmHg	63.6 ± 8.6	67.0 ± 9.9 **	70.1 ± 9.4 [a]	77.2 ± 12.2 **[a]

Comorbidities were arterial hypertension, type 2 diabetes and dyslipidemia.
•1-repetition maximum strength test was adjusted for body mass. SBP, systolic blood pressure; DBP, diastolic blood pressure. Different from normal-weight at the same comorbidity group(*P<0.05; **P<0.01; and***P<0.0001). Different from without comorbidities at the same BMI group ([a]P<0.05).

Table 4 - Muscle strength, cardiorespiratory fitness, heart rate and blood pressure responses to exercise before and after 12 months of exercise training.

	Normal-weight (n = 45)		Overweight/Obese (n = 39)	
Characteristics	**Pre**	**Post**	**Pre**	**Post**
1- RM Test •				
Upper extremity, $kg \cdot kg^{-0.67}$	9.38 ± 1.32	12.47 ± 2.06 ‡	8.17 ± 1.62 *	11.56 ± 1.68 *‡
Lower extremity, $kg \cdot kg^{-0.67}$	10.12 ± 2.76	15.13 ± 3.06 ‡	8.73 ± 2.39 *	12.9 ± 3.01 *‡
GXT				
Exercise capacity, METs	10.00 ± 0.81	10.75 ± 0.72 †	8.80 ± 0.92 **	9.20 ± 1.21 **
Duration, min	9.48 ± 1.58	10.41 ± 1.13 †	8.43 ± 1.42 **	9.19 ± 1.20 **
$HR_{RESTING}$, bpm	85.0 ± 17.6	79.8 ± 11.4	84.9 ± 11.2	79.6 ± 9.0
HR_{PEAK}, bpm	177.8 ± 9.5	176.0 ± 9.6	172.5 ± 10.6 *	169.7 ± 11.8 *
% of predicted HR_{PEAK}	104.2 ± 4.7	104.4 ± 6.2	101.4 ± 6.3 *	99.8 ± 6.2 *
$HR_{RESERVE}$, bpm	92.8 ± 10.6	96.2 ± 11.8	87.6 ± 10.8	90.1 ± 13.1
% of predicted $HR_{RESERVE}$	109.7 ± 9.7	108.0 ± 10.1	102.8 ± 13.3 *	99.4 ± 12.4 *
$HR_{RECOVERY}$, beats	19.9 ± 6.9	25.7 ± 7.9 †	17.9 ± 9.9	20.9 ± 9.2
Blood Pressure				
SBP_{REST}, mmHg	112.6 ± 19.0	112.5 ± 22.2	122.8 ± 22.8	114.7 ± 15.5
DBP_{REST}, mmHg	70.5 ± 11.1	71.6 ± 11.1	79.1 ± 22.3	73.3 ± 9.8
SBP_{PEAK}, mmHg	148.3 ± 17.2	151.9 ± 18.2	169.8 ± 20.9 **	158.9 ± 16.8
DBP_{PEAK}, mmHg	67.3 ± 12.1	67.5 ± 11.1	76.6 ± 13.9	69.5 ± 9.7
$SPB_{RECOVERY}$, mmHg	130.2 ± 25.5	128.1 ± 19.3	151.0 ± 20.5 *	148.6 ± 17.3 *
$DBP_{RECOVERY}$, mmHg	71.2 ± 16.2	67.4 ± 10.4	73.0 ± 8.9	69.8 ± 8.7

•1-repetition maximum strength test was adjusted for body mass. SBP, systolic blood pressure; DBP, diastolic blood pressure. Different from normal-weight women at the same period (*P<0.05; **P<0.01; and ***P<0.001). Different from pre-exercise in the same group (†p<0.05 and ‡p<0.001). 1-RM, 1-repetition maximum strengh; GXT, graded exercise test; HR, heart rate.

systems adapt similarly to exercise training in normal-weight and overweight/obese subjects.

In the present study, normal-weight and overweight/obese women displayed similar upper and lower extremity muscle strength responses to exercise, which are findings that have been confirmed by several investigators[26,27], though not by all studies.[28,29] Discrepancies between the previous studies may be due to differences in the resistance training intervention. These discrepancies includes lower exercise training volume[28,29] and intensity[28], which may prevent some adaptations that result from the whole body resistance training program used in the present study and others.[26,27] In contrast to the similarities in exercise-induced, muscle-strength adaptations, overweight/obese women displayed an impaired CRF increase (4.4%, P = 0.52) in response to exercise compared with normal-weight women (6.6%, P = 0.049). These data conflict with previous studies that report a 12-22% increase in CRF in overweight/obese subjects following an exercise training program[18,19]; however, these previous studies did not include a normal-weight group for comparison. The present study showed that overweight/obese postmenopausal women attain a lower exercise-induced CRF increase than normal-weight postmenopausal women following the same exercise program. The increased CRF adaptation observed in previous studies may be explained by higher exercise intensity[18] and volume[18,19] of the training. Similar CRF changes (4.2%) than the observed in present study were noted in a study using overweight/obese women following a thrice weekly, 30-minute, moderate-intensity exercise program.[17]

In addition, ANS activity is also impaired in overweight/obese individuals.[8-11] To evaluate ANS activity, we used the GXT, which is an easy and inexpensive tool that provides a wealth of information regarding the interactions between the ANS and cardiovascular system at various phases of rest, exercise and recovery[12,13]. We found that peak HR (absolute and relative) and reserve HR (relative), but not resting and recovery HR, were reduced in overweight/obese women. These findings are in line with a previous study showing similar resting and lower peak HRs in overweight/obese subjects compared to normal-weight individuals.[41] In contrast, exercise training improved recovery HR (6 bpm; P = 0.02) in normal-weight women but not in overweight/obese women. The resting HR was reduced similarly (5 bpm) in both groups; however, this reduction failed to achieve significance. These findings conflict with previous studies that showed exercise training-induced improvements in baroreflex sensitivity and parasympathetic activity (increased high-frequency spectral power of HR variability) following acute exercise[11] and improved resting ANS activity (HR variability) in overweight/obese subjects.[10] However, similar to the studies showing exercise-induced improvements in CRF,[18,19] these studies did not include a normal-weight group for comparison.

In accordance with the present findings, a cross-sectional study did not show any differences in resting, peak or recovery HR between sedentary and trained obese subjects with impaired peak and recovery HR compared to normal-weight subjects.[41] The initial increase in HR during exercise is caused by a withdrawal of the vagal tone followed by an increase in sympathetic activation, which further increases during exercise. Recovery HR is mainly related to an increase in vagal tone, which occurs immediately after exercise cessation[14]. The lack of improvement in the recovery HR in the overweight/obese group compared to the normal-weight group in the present study suggests a blunted exercise-induced improvement in parasympathetic activity in the overweight/obese postmenopausal women. The discrepancies in the response of the ANS to exercise training between previous studies[10,11] and the present study

may be due to differences in the methods used to analyze ANS activity (baroreflex sensitivity and HR variability x HR response to GXT) and differences in the time that ANS activity was measured (resting or following a submaximal exercise x during a GXT).

The reductions in systolic and diastolic BP responses to exercise were observed only in overweight/obese women, and these data agree with previous studies in which exercise-induced BP improvements occurred only in poorly BP-controlled subjects.[42-44] Because of the small population studied, these BP reductions cannot be dismissed because of the absence of statistical significance, and they confirm the positive effects of exercise training on BP control.[16,42-45]

Impairments in HR increase, chronotropic index, HR recovery during exercise,[12,13] and low CRF[6] are strong and independent predictors of sudden death. Therefore, the reduced baseline peak and reserve HR (indicators of chronotropic index) and the blunted exercise-induced improvements in CRF and recovery HR observed in overweight/obese postmenopausal women have important implications.

The present study has several limitations. Because exercise training is recommended for 5 or more days per week for obese subjects[15] to achieve the greatest improvements in CRF,[18,19] the frequency of training performed (3 times per week) in the present study may be a limitation. It is not known whether the impaired exercise-induced adaptation in the CRF and HR responses to exercise in the overweight/obese group would be observed with a greater volume and/or frequency of training. However, it is important to emphasize that exercise training of similar intensity and frequency has been shown to improve several health and fitness variables.[11,17,18,33] In addition, the exercise protocol was identical for every subject in both groups. We did not directly measure oxygen consumption because this is not a component of our routine exercise stress test. However, the absence of an exercise-induced increase in CRF (METs) in overweight/obese women was in line with the absence of an exercise-induced increase in exercise time during GXT, another important indicator of CRF.[30]

In summary, there were no differences in the exercise-induced muscle strength increases between normal-weight and overweight/obese postmenopausal women. However, overweight/obese women displayed an impaired HR response to exercise, and only normal-weight women improved CRF and recovery HR after exercise training. These results suggest that exercise-induced improvements in CRF and HR response to exercise may be impaired in overweight/obese postmenopausal women.

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